Evaluation of Analgesic, Anti-Inflammatory and CNS Depression Activities of the Methanolic Extract of *Acacia nilotica* Seed

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**Abstract:** The present study was designed to evaluate the analgesic, anti-inflammatory and CNS depression activities of the methanolic extract of *Acacia nilotica* seed. *Acacia nilotica* seed (ANS) and seed pods (ANSP) were evaluated for anti-inflammatory activity using carrageenan induced hind paw edema model whereas analgesic activity was evaluated by acetic acid induced writhing and formalin induced linking tests and hole cross and open field tests were considered to evaluate CNS depression activity. ANS and ANSP, at the dose/doses of 150 and 300 mg/kg, significantly \(p<0.05\) reduced the writhing caused by acetic acid and the number of licks induced by formalin in a dose dependent manner. The same ranges of doses of ANS and ANSP caused significant \(p<0.05\) inhibition of carrageenan induced paw edema after 4 h in a dose dependent manner. A statically significantly decrease in locomotor activity at doses of 150 and 300 mg/kg was also observed in both hole cross and open field tests. The findings of the study suggested that *Acacia nilotica* seed has strong analgesic, anti-inflammatory and CNS effects, conforming the traditional use of this plant.

**Key words:** Analgesic • Anti-Inflammatory • CNS Activity • *Acacia nilotica*

**INTRODUCTION**

At present, lots of plant secondary metabolites where 35% are considered as drugs and are successfully used for the ailments of various diseases [1]. The investigation of the efficacy of plant-based drugs used in the traditional medicine has been paid great attention because they are cheap, have little side effects [2]. In Bangladesh thousands of species are known to have medicinal value and the use of different parts of several medicinal plants to cure specific ailments has been in vogue since ancient times.

*Acacia nilotica* belongs to the family of Fabaceae, popularly regarded as a medicinal plant in the South East Asian region. Experimentally, *Acacia nilotica* is shown to possess antibacterial, anti-inflammatory [3], Antimutagenic [4], kinase inhibitor [5], free radical scavenging [6,7], Anti-helminthic [8], Anti quorum sensing [7], larvicidal activity [9], cytotoxic effects [10] dose related contractile activity [11], anti spasmodic and antihypertensive activity [12] and molluscidal activity [13]. Leaf and bark infusions have diuretic, anti-inflammatory and cicatrizant uses [14]. The chief constituent of *Acacia nilotica* have/has been reported alkaloids, glycosides, cyclitols, fatty acids, terpenes, hydrolysable tannins, flavonoids and condensed tannins [14]. It is also rich in phenolics consisting of condensed tannin and phlobatannin, gallic acid, protocatechuic acid pyrocate, (+)–catechin, (-)epigallocatechin-7-gallate and (-)epigallocatechin-5, 7-digallate [15]. Literature reviews indicated that no combined studies in analgesic, anti-inflammatory and CNS depression effects of the seed of *Acacia nilotica* have so far been undertaken. Taking this in view and as a part of our ongoing research [16] on Bangladeshi medicinal plants, the present study was aimed to evaluate the analgesic, anti-inflammatory and CNS depression activities of the methanolic extract of the seeds of *Acacia nilotica* in different experimental model.
MATERIALS AND METHODS

Plant Materials: Acacia nilotica was collected from the adjoining area of Dattanager, Rajshahi, Bangladesh during February 2012. The plant material was taxonomically identified by the National Herbarium of Bangladesh and voucher specimen was maintained in our laboratory for future reference.

Chemicals: Carrageenan was purchased from E. Merck (Germany). Nalbuphine, Indomethacin, Diclofenac-Na was collected from Square Pharmaceuticals Ltd., Bangladesh. All other chemicals and reagents were of analytical grade.

Preparation of Plant Extract: The both plant materials were shade-dried with occasional shifting and then powdered with a mechanical grinder, passing through sieve#40 and stored in a tight container. The dried powder material of seed (1.2 kg) and seed pod (1.0 kg) was refluxed with MeOH for three hours. The total filtrate was concentrated to dryness, in vacuo at 40°C to render the MeOH extract 200 g and 180 g for seed and seed pod respectively.

Animal: Young Long-Evans rats of either sex weighing about 80-120gm and Swiss albino mice (25-30g) were used for assessing biological activity. The animals were maintained under standard laboratory conditions and had free access to food and water ad libitum. The animals were allowed to acclimatize to the environment for 7 days prior to experimental session. The animals were divided into different groups, each consisting of five animals which were fasted overnight prior to the experiments. Experiments on animals were performed in accordance with guidelines of the Institutional Animal Ethics Committee, Atish Dipankar University of Science & Technology, Dhaka, Bangladesh. Animal treatment and maintenance for acute toxicity and analgesic effects were conducted in accordance with the Principle of Laboratory Animal Care (NIH publication No. 85-23, revised 1985) and the Animal Care and Use Guidelines of Atish Dipankar University of Science & Technology, Dhaka, Bangladesh.

Acute Toxicity Study: Acute oral toxicity assay was performed in healthy nulliparous and non pregnant adult female albino Swiss mice (25-30g) divided into different groups. The test was performed using increasing oral dose of the ANS and ANSP (50, 100, 200, 500, 1000 mg/kg body weight), in 20 ml/kg volume to different test groups. Normal group received water. The mice were allowed to feed ad libitum, kept under regular observation for 48 hrs, for any mortality or behavioral changes [17].

Analgesic Activity
Acetic Acid-Induced Writhing Test: The analgesic activity of the samples was studied using acetic acid-induced writhing model in mice. The animals were divided into six groups with five mice in each group. Group I animals received vehicle (1% Tween 80 in water, 10 ml/kg body weight), animals of Group II received Diclofenac-Na at 10 mg/kg body weight while animals of groups III, IV, V and VI were treated with 150 and 300 mg/kg body weight (p.o.) of the seed and seed pods. Test samples and vehicle were administered orally 30 min before intraperitoneal administration of 0.7% v/v acetic acid but Diclofenac-Na was administered intraperitoneally 15 min before injection of acetic acid. After an interval of 5 min, the mice were observed for specific contraction of body referred to as ‘writhing’ for the next 10 min [18].

Formalin Test: The antinociceptive activity of the drugs was determined using the formalin test described by Dubuisson and Dennis [19]. Control group received 5% formalin. 20 µl of 5% formalin was injected into the dorsal surface of the right hind paw 30 min after administration of ANS and ANSP (150 and 300 mg/kg, p.o.) and 15 min after administration of Diclofenac Na (10 mg/kg, i.p.). The mice were observed for 30 min after the injection of formalin and the amount of time spent licking the injected hind paw was recorded. The first 5 min post formalin injection is referred to as the early phase and the period between 15 and 30 min as the late phase. The total time spent licking or biting the injured paw (pain behavior) was measured with a stop watch.

Anti-Inflammatory Activity: Long-Evan rats (100-120 g) of both sexes were divided into six groups of five animals each. The test groups received 150 and 300 mg/kg body weight, p.o. of the extract ANS and ANSP. The reference group received indomethacin (10 mg/kg body weight, p.o.) while the control group received 3 ml/kg body weight normal saline. After 30 min, 0.1 ml 1% carrageenan suspension in normal saline was injected into the subplantar tissue of the right hind paw. The paw volume was measured at 1, 2, 3 and 4 hrs after carrageenan injection using a micrometer screw gauge. The percentage inhibition of the inflammation was calculated from the formula:
% inhibition = (1 - D, D, x 100

where, D, was the average inflammation (hind paw edema) of the control group of mice at a given time, D, was the average inflammation of the drug treated (i.e., extract or reference Indomethacin) mice at the same time [20].

CNS Depressant Activity

**Hole Cross Test:** The method used was done as described by Takagi et al. [21]. The animals were divided into different groups and each group contains 6 animals. The control group received vehicle (1% Tween 80 in water at the dose of 10 ml/kg, p.o.) whereas the test group received the crude extract (At the doses of 150 and 300 mg/kg, p.o.) and standard group received Diazepam at the dose of 1mg/kg body weight orally. Each animal was then placed on one side of the chamber and the number of passages of each animal through the hole from one chamber to the other was recorded for 3 min on 0, 30, 60, 90 and 120 min during the study period.

**Open Field Test:** This experiment was carried out as described by Gupta et al. [22]. The animals were divided into control standard and test groups (N = 6 per group). The control group received vehicle (1% Tween 80 in water at the dose of 10 ml/kg, p.o.). The test group received the crude extract (At the doses of 250 and 500 mg/kg p.o.) and standard group received Diazepam at the dose of 1mg/kg body weight orally. The animals were placed on the floor of an open field (100 cm x 100 cm x 40 cm h) divided into a series of squares. The number of squares visited by each animal was counted for 3 min on 0, 30, 60, 90, 120, 180 and 240 min during the study period.

**Statistical Analysis:** All values were expressed as the mean ± SEM of three replicate experiments. The analysis was performed by using SPSS statistical package for WINDOWS (Version 16.0; SPSS Inc, Chicago). Results related to the reducing power activities were statistically analyzed by applying the Student t-test andp < 0.001 were considered to be statistically significant. All in vivo data are subjected to ANOVA followed by Dunnett’s test andp < 0.05 were considered to be statistically significant.

**RESULTS**

**Acute Toxicity Studies:** The acute toxicity studies mainly aim at establishing the therapeutic index, i.e., the ratio between the pharmacologically effective dose and the lethal dose on the same strain and species. Both extract of ANS and ANSP were safe up to a dose of 1000 mg/kg (P.o.) body weight. Behavior of the animals was closely observed for the first 3h then at an interval of every 4h during the next 48h. The extract did not cause mortality in mice during 48h observation but little behavioral changes, locomotor ataxia, diarrhoea and weight loss were observed. Food and water intake had no significant difference among the group studied.

**Analgesic Activity**

**Acetic Acid-Induced Writhing Test:** Table 1 shows the effects of both extract of on acetic acid-induced writhing in mice. The oral administration of both doses of ANS and ANSP significantly (P<0.05) inhibited writhing response induced by acetic acid in a dose dependent manner.

**Formalin Test:** ANS and ANSP (150 and 300 mg/kg, p.o.) significantly (P<0.05) suppressed the licking activity in either phase of the formalin-induced pain in mice in a dose dependant manner (Table 2). ANS and ANSP, at the dose of 300 mg/kg body weight, showed the almost similar licking activity against both phases of formalin-induced pain than that of the standard drug Diclofenac-Na.

**Antibody Activity:** Figure 1 shows the results of the anti-ematous effects of orally administered of ANS and ANSP on carrageenan induced paw edema in mice. Both extract showed dose dependent anti-inflammatory activity and statistically significant (P<0.05). At 300 mg/kg dose both ANS and ANSP showed remarkable anti-inflammatory effects (68.86% and 60.19% inhibition, respectively), whereas standard Indomethacin showed 77.72% of inhibition of paw edema.

**CNS Depression Activity**

**Hole-Cross Test:** In the Hole-cross test, ANS and ANSP extracts exhibited a decrease in the movements of the test animals at all dose levels tested. They were statistically significant (P<0.05) for all dose levels and followed a dose-dependent response. The depressing effect was most intense during the second (60 min) and third (90 min) observation periods in both extract (Table 3).

**Open-Field Test:** In the open-field test, both ANS and ANSP extract exhibited a decrease in the movements of the test animals at all dose levels tested. The results were statistically significant for all doses and followed a dose-dependent response (Table 4).
Table 1: Effects of methanolic extract of ANS and ANSP on acetic acid-induced writhing in mice

<table>
<thead>
<tr>
<th>Groups</th>
<th>Dose (mg/kg)</th>
<th>No. of writhing</th>
<th>% inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>Vehicle</td>
<td>30.67 ± 1.10</td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>10</td>
<td>8.50 ± 1.30*</td>
<td>72.28</td>
</tr>
<tr>
<td>Group III</td>
<td>150</td>
<td>20.00 ± 1.86</td>
<td>34.78</td>
</tr>
<tr>
<td>Group IV</td>
<td>300</td>
<td>11.16 ± 2.06*</td>
<td>63.61</td>
</tr>
<tr>
<td>Group V</td>
<td>150</td>
<td>25.12 ± 2.06</td>
<td>18.09</td>
</tr>
<tr>
<td>Group VI</td>
<td>300</td>
<td>18.00 ± 1.24*</td>
<td>41.31</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, (n = 5), *P<0.05 as compared to vehicle control (One way ANOVA followed by Dunnet test). Group I animals received vehicle (1% Tween 80 in water), Group II received Diclofenac Na 10 mg/kg body weight, groups III, IV, V and VI were treated with 150 and 300 mg/kg body weight (p.o.) of ANS and ANSP respectively.

Table 2: Effects of methanolic extract of ANS and ANSP in the hindpaw licking in the formalin test in mice

<table>
<thead>
<tr>
<th>Groups</th>
<th>Dose (mg/kg)</th>
<th>Early phase (Sec)</th>
<th>% protection</th>
<th>Late phase (Sec)</th>
<th>% protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group-I</td>
<td>Vehicle</td>
<td>35.0 ±1.89</td>
<td>-</td>
<td>43.66 ± 1.82</td>
<td>-</td>
</tr>
<tr>
<td>Group-II</td>
<td>10</td>
<td>12.00 ±1.23*</td>
<td>65.71</td>
<td>12.13 ± 0.36*</td>
<td>72.21</td>
</tr>
<tr>
<td>Group-III</td>
<td>150</td>
<td>25.50±1.89</td>
<td>27.14</td>
<td>23.16 ± 1.82</td>
<td>46.95</td>
</tr>
<tr>
<td>Group-IV</td>
<td>300</td>
<td>12.50 ±1.69*</td>
<td>64.28</td>
<td>10.83 ± 1.52*</td>
<td>75.19</td>
</tr>
<tr>
<td>Group-V</td>
<td>150</td>
<td>29.20 ± 1.89</td>
<td>16.57</td>
<td>25.96 ± 1.82</td>
<td>40.54</td>
</tr>
<tr>
<td>Group VI</td>
<td>300</td>
<td>15.0 ±1.12*</td>
<td>57.14</td>
<td>11.16 ± 1.99*</td>
<td>74.34</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, (n = 5), *P<0.05 as compared to vehicle control (One way ANOVA followed by Dunnet test). Group I animals received vehicle (1% Tween 80 in water), Group II received Diclofenac Na 10 mg/kg body weight, groups III, IV, V and VI were treated with 150 and 300 mg/kg body weight (p.o.) of ANS and ANSP respectively.

Table 3: Effect of methanolic extract of the ANS and ANSP on hole cross test in mice

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose</th>
<th>0 min</th>
<th>30 min</th>
<th>60 min</th>
<th>90 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group-I</td>
<td>10ml/kg,</td>
<td>11.6± 1.35</td>
<td>17 ± 1.7</td>
<td>17.2 ± 0.96</td>
<td>18.6 ± 1.2</td>
</tr>
<tr>
<td>Group-II</td>
<td>1mg/kg,</td>
<td>15 ± 1.1</td>
<td>5.0± 0.61*</td>
<td>3.4± 0.57*</td>
<td>2.6± 3.44*</td>
</tr>
<tr>
<td>Group-III</td>
<td>150 mg/kg</td>
<td>3.8 ± 1.5*</td>
<td>2.4±.57*</td>
<td>3.2± 0.65*</td>
<td>1.8± 0.41*</td>
</tr>
<tr>
<td>Group-IV</td>
<td>300 mg/kg</td>
<td>2.2 ± 0.65*</td>
<td>5.8±0.65*</td>
<td>5.0±0.5*</td>
<td>2.1± 0.02*</td>
</tr>
<tr>
<td>Group-V</td>
<td>150 mg/kg</td>
<td>3.00±0.61*</td>
<td>1.6±0.44*</td>
<td>1.4±0.44*</td>
<td>1.0±0.02*</td>
</tr>
<tr>
<td>Group VI</td>
<td>300 mg/kg</td>
<td>1.00± 0.23*</td>
<td>2.4±0.44*</td>
<td>2.4± 0.44*</td>
<td>1.8±0.65*</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, (n = 6); *p<0.05, Dunnet test as compared to vehicle control. Group I animals received vehicle (1% Tween 80 in water), Group II received Diazepam 1 mg/kg body weight, groups III, IV, V and VI were treated with 150 and 300 mg/kg body weight (p.o.) of the ANS and ANSP, respectively.

Table 4: Effect of methanolic extract of the ANS and ANSP on open field test in mice

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose</th>
<th>0 min</th>
<th>30 min</th>
<th>60 min</th>
<th>90 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group-I</td>
<td>10ml/kg,</td>
<td>118.4 ± 1.20</td>
<td>118 ± 1.30</td>
<td>115.4 ± 0.50</td>
<td>117.4 ± 1.16</td>
</tr>
<tr>
<td>Group-II</td>
<td>1mg/kg,</td>
<td>117.2 ± 1.15</td>
<td>64.6±0.43*</td>
<td>40.8± 0.58*</td>
<td>18.8± 0.86*</td>
</tr>
<tr>
<td>Group-III</td>
<td>150 mg/kg</td>
<td>110.4 ± 0.81</td>
<td>70.8±1.02*</td>
<td>47.8±1.35*</td>
<td>32.8± 0.02*</td>
</tr>
<tr>
<td>Group-IV</td>
<td>300 mg/kg</td>
<td>117.8 ± 1.43</td>
<td>60.2±0.06*</td>
<td>41.6±0.92*</td>
<td>21.6±0.92*</td>
</tr>
<tr>
<td>Group-V</td>
<td>150 mg/kg</td>
<td>121.4 ± 1.81</td>
<td>79.5±1.12*</td>
<td>51.8±1.15*</td>
<td>40.2± 0.62*</td>
</tr>
<tr>
<td>Group VI</td>
<td>300 mg/kg</td>
<td>107.8 ± 1.13</td>
<td>71.9±0.66*</td>
<td>45.6±0.42*</td>
<td>37.9±0.22*</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, (n = 6); *p<0.05, Dunnet test as compared to vehicle control. Group I animals received vehicle (1% Tween 80 in water), Group II received Diazepam 1 mg/kg body weight, groups III, IV, V and VI were treated with 150 and 300 mg/kg body weight (p.o.) of the ANS and ANSP, respectively.
DISCUSSION

Acetic acid induced writhing response is a sensitive procedure to evaluate peripherally acting analgesics and represents pain sensation by triggering localized inflammatory response. Such pain stimulus leads to the release of free arachidonic acid from the tissue phospholipid [23]. The response is thought to be mediated by peritoneal mast cells [24], acid sensing ion channels [25] and the prostaglandin pathways [26]. The organic acid has also been postulated to act indirectly by inducing the release of endogenous mediators, which stimulates the nociceptive neurons that are sensitive to NSAIDs and narcotics [16]. It is well known that non-steroidal anti-inflammatory and analgesic drugs mitigate the inflammatory pain by inhibiting the formation of pain mediators at the peripheral target sites where prostaglandins and bradykinin are proposed to play a significant role in the pain process [27]. Therefore, it is likely that ANS and ANSP might have exerted its peripheral antinociceptive action by interfering with the local reaction caused by the irritant or by inhibiting the synthesis, release and/or antagonizing the action of pain mediators at the target sites. The above findings clearly demonstrated that both central and peripheral mechanisms are involved in the antinociceptive action of ANS and ANSP. Interestingly, compounds like flavonoids and steroids, triterpenes in part, have been shown to possess anti-inflammatory, analgesic activity and the claim made by Pritam et al. [28].

The formalin model normally postulates the site and the mechanism of action of the analgesic. This biphasic model is represented by neurogenic (0-5 min) and inflammatory pain (15-30 min), respectively [29]. Drugs that act primarily on the central nervous system such as narcotics inhibit both as steroids and NSAIDs suppress mainly the late phase [16]. The suppression of neurogenic and inflammatory pains by the extract might imply that it contains active analgesic principle that may be acting both centrally and peripherally. This is an indication that the extract can be used to manage acute as well as chronic pain. The mechanism by which formalin triggers C-fibers activation remained unknown for a relatively long time. Recently, however, McNamara et al. [30] demonstrated that formalin activates primary afferent neurons through a specific and direct on TRPA1, a member of the transient receptor potential family of cation channels, expressed by a subset of C-fiber nociceptors and this effect is accompanied by increased influx of Ca$^{2+}$ ions. TRPA1 cation channels at primary sensory terminals were also reported to mediate noxious mechanical stimuli [31]. These experiments suggest that Ca$^{2+}$ mobilization through TRPA1 cation channels is concomitant with noxious chemicals and mechanical stimuli as they produce their
analgesic action. It is likely that the inhibitory effect of 
ANS and ANSP to pain response is due to inhibit the 
increase of the intracellular Ca\(^{2+}\) through TRPA1, 
previously evoked by formalin. So, ANS and ANSP 
may contain substances that affect the metabolism of 
Ca\(^{2+}\).

Carrageenan induced edema has been commonly 
used as an experimental animal model for acute 
inflammation and is believed to be biphasic. The early 
phase (1-2hrs) of the carrageenan model is mainly 
mediated by histamine, serotonin and increased synthesis 
of prostaglandins in the damaged tissue surroundings. 
The late phase is sustained by prostaglandin release and 
mediated by bradykinin, leukotrienes, polymorphonuclear 
cells and prostaglandins produced by tissue macrophages 
[32]. Since the extract significantly inhibited paw edema 
induced by carrageenan in the second phase and this 
finding suggests a possible inhibition of cyclooxygenase 
synthesis by the extract and this effect is similar to that 
produced by non-steroidal anti-inflammatory drugs such 
as Indomethacin, whose mechanism of action is inhibition 
of the cyclooxygenase enzyme. Flavonoids and saponins 
are well known for their ability to inhibit pain perception 
as well as anti-inflammatory properties due to their 
inhibitory effects on enzymes involved in the production 
of the chemical mediator of inflammation. This hypothesis 
is strongly supported by the previous study, which has 
shown that Acacia nilotica possess anti-inflammatory 
activity due to the presence of high flavonoid content 
[33].

Locomotor activity considered as an increase in 
alertness and decrease in locomotor activity indicated 
sedative effect [34]. Extracts of ANS and ANSP decreased 
locomotor activity indicates its CNS depressant activity. 
Gamma-aminobutyric acid (GABA) is the major inhibitory 
neurotransmitter in the central nervous system. Different 
anxiolytic, muscle relaxant, sedative-hypnotic drugs are 
elucidation their action through GABA, therefore it is 
possible that extracts of ANS and ANSP may act by 
potentiation GABAergic inhibition in the CNS via 
membrane hyperpolarization which lead to a decrease in 
the firing rate of critical neurons in the brain or may be 
due to direct activation [33] on of GABA receptor by the 
extracts (Many research showed that plant containing 
flavonoids, saponins and tannins are useful in many CNS 
disorders [35]. Earlier investigation on phytoconstituents 
and plants suggests that many flavonoids and 
neuroactive steroids were found to be ligands for the 
GABA\(_A\) receptors in the central nervous system; which 
led to assume that they can act as Benzodiazepine like 
molecules [34]. Phytochemical investigations also showed 
the presence of alkaloids, flavonoids and tannins in the 
extract, so might be this phyto-constituents are 
responsible for its CNS depressant activity.

**CONCLUSION**

The results of the experiments suggest that 
Acacia nilotica may be used as an alternative or 
supplementary herbal remedy for the treatment of 
analgesic and inflammatory disease. Because of its 
algesic and anti-inflammatory effects, may have 
Acacia nilotica beneficial effects together with drugs 
known for a strong analgesic as well as anti-inflammatory 
effects. Thus the present study warrants further 
investigation involving components of Acacia nilotica 
for possible development of new class of analgesic and 
anti-inflammatory drugs.

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